

77. (Previously Added) The mammalian host cell of claim 76, further comprising an AAV virion, viral particle, or an rAAV vector.
78. (Previously Added) The mammalian host cell of claim 77, wherein said AAV virion, viral particle, or rAAV vector comprises a nucleic acid segment that encodes a therapeutic polypeptide.

## 1.2 IN THE SPECIFICATION:

Please replace the paragraph on page 44, lines 2-21, with the following:

HSV-1 (wt KOS strain) was propagated by infecting Vero cells (90% confluent in T175 flasks) at a multiplicity of infection (MOI) of 0.1 per cell. Adsorption of virus was done for 45 min in reduced serum DMEM (2% FCS). After full cytopathic effect (CPE) was observed (usually 48 h post infection) the cell pellet was collected by centrifugation (1000 rpm for 10 min), then frozen and thawed 3 times. Cell debris was removed by centrifugation (3000 rpm for 5 min). d27-1 is an ICP27 deletion of HSV-1 (KOS strain) and has been previously described (Rice and Knipe, 1990). d27-1 (ATCC PTA-4004) was propagated as described for HSV-1 except that the complementing cell line, V27 (ATCC PTA-4296), was used. Ad5 (from the *American Type Culture Collection*, Rockville, MD) was propagated by infecting 293 cells (90% confluent in 15 cm dishes) at an MOI of 0.1 per cell. Ad5 was harvested as described for HSV-1 after full CPE was observed (usually 72 to 96 h post infection). AAV-2 was propagated by coinfection of 293 cells with AAV-2 (MOI of 200 particles per cell) and Ad5 (MOI of 0.1). AAV-2 viral lysates were prepared by freeze-thaw, and the Ad5 was heat inactivated by

incubation at 55°C for 45 min. HSV-1 (wt KOS) was titered by plaque forming assay on Vero cells. d27-1 was titered by plaque forming assay on V27 cells. Analysis of d27-1 stocks for the presence of wt HSV-1 was done by plaque assay on non-complementing Vero cells (<100 pfu/ml detected). Ad5 was titered by plaque forming assay on 293 cells. AAV-2 was titered for particles by dot blot analysis as described below for recombinant genomes in the amplicon stocks.

Please insert the following paragraphs after line 21 on page 44:

“Subject cultures of the invention have been deposited under conditions that assure that access to the cultures will be available during the pendency of this patent application to one determined by the Commissioner of Patents and Trademarks to be entitled thereto under 37 C. F. R. §§1.14 and 1.801 and 35 U. S. C. §122. The deposits are available as required by foreign patent laws in countries wherein counterparts of the subject application, or its progeny, are filed. However, it should be understood that the availability of a deposit does not constitute a license to practice the subject invention in derogation of patent rights granted by governmental action.

The subject culture deposits will be stored and made available to the public in accord with the provisions of the Budapest Treaty for the Deposit of Microorganisms, i.e., they will be stored with all the care necessary to keep them viable and uncontaminated for a period of at least five years after the most recent request for the finishing of a sample of the deposit, and in any case, for a period of at least 30 (thirty) years after the date of deposit or for the enforceable life of any patent which may issue disclosing the cultures. The depositor acknowledges the duty to replace the deposits should the depository be unable to furnish a sample when requested, due to the condition of the deposits. All restrictions on the availability to the public of the subject culture deposits will be irrevocably removed upon the granting of any patent disclosing them.

Recombinant herpes simplex virus rHSV d27.1rc was deposited February 1, 2002, and the host cell line V27 was deposited May 7, 2002 in the permanent collection of the American Type Culture Collection, 10801 University Blvd., Manassas, VA, 20110-2209, USA, under the terms of the Budapest Treaty. The required certificate of viability for rHSV d27.1 was issued by